EDITORIAL

A cardiologist's guide to waist management

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Clinical management of obesity remains a challenge

Obesity results from a complex interplay of genetic predisposition and environmental factors that operate throughout an individual's lifetime. While obesity has a significant negative impact on nearly all aspects of health, its importance as a risk factor for cardiovascular diseases (CVD) stands out as the leading determinant of obesity-related morbidity and mortality.[1] The atherosclerotic and thrombotic vascular diseases, commonly associated with obesity, are heterogeneous entities that result from the concurrent haemodynamic, inflammatory, prothrombotic, and metabolic derangements resulting from an unhealthy body weight.

IMPORTANCE OF ABDOMINAL OBESITY

Traditionally, obesity has been defined using body mass index (BMI).[2] However, BMI does not discriminate between muscle and fat mass, or provide a measure of fat distribution, the key determinant of cardiometabolic risk related to excess body weight. In INTERHEART we studied the relationship of BMI and waist-to-hip ratio (WHR) to the risk of myocardial infarction (MI) using data from about 15 000 cases and a similar number of controls representing multiple ethnic groups.[3] INTERHEART demonstrates that the WHR is the strongest anthropometric measure associated with MI risk, and is notably superior to BMI. Indeed, even at the lowest level of BMI, increased WHR still notably increases the risk for MI. In contrast, individuals with high BMI but low WHR do not show excess risk. These results are consistent in both sexes, old and young individuals, in different regions and among ethnic groups. If we used elevated WHR as the index of obesity instead of BMI, the population attributable risk of MI due to excess body weight would triple.

There are important morphological and functional differences between visceral and subcutaneous adipose tissue,[4] which may account for the increased cardiovascular risk associated with increased abdominal fat. Visceral adipocytes are generally smaller and more lipolytically active than subcutaneous adipocytes, thereby exposing the liver to a higher concentration of free fatty acids. Visceral adipose tissue generates greater quantities of angiotensinogen, plasminogen activator inhibitor-1, tumour necrosis factor-α, and resistin, and less leptin and adiponectin. It is therefore not surprising that several studies have demonstrated that reduction in waist circumference by weight loss is associated with pronounced improvements in both inflammatory markers as well as metabolic parameters and cardiovascular function.[5] In contrast, the surprisingly protective effect of large hips is relatively unclear. This protective effect may be due to biological characteristics of gluteal fat (as a fat sink) but could also be attributable to the fact that hip circumference may be an indirect measure of gluteal muscle mass—a surrogate for lean body mass.

LIFESTYLE MANAGEMENT OF ABDOMINAL OBESITY

The factors leading to increased excess body fat operate at societal (urbanisation, automation), economic (cost of foods and leisure time activities), cultural (attitudes towards food and activity), and individual (food choices, leisure activities) levels.[6] Sedentary living associated with extensive screen time is a key determinant of increased abdominal fat deposition, but dietary factors may also be important.[7] Thus, higher intakes of protein (even modest amounts)[8] and fibre[7] in isocaloric diets are associated with reduced central adiposity. Eating half a chicken breast instead of 150 g of cooked rice (approximately one cup) equals a 30 g substitution of protein for carbohydrate intake, and would be associated with a 1.5 unit reduction in percentage WHR.[8] Eucaloric substitution of polyunsaturated fat for saturated and trans fats in the diet is associated with less central adiposity.[7] Increasing vigorous physical activity by 25 metabolic equivalent tasks (METs) h/week and $\geqslant$ 0.5 h/week in weight training were associated with 0.38 cm and 0.91 cm decreases in waist circumference, respectively ($p < 0.001$ for each comparison).[7] These associations remained significant after further adjustment for BMI. Although smoking is associated with reduced BMI, it may be associated with increased central adiposity. There was an approximately 2 unit increase in percentage WHR in current smokers compared with never smokers holding BMI constant.[8] Smoking increases interleukin-6 and tumour necrosis factor-α values[9] and insulin resistance.[10] Higher cytokine concentrations are associated with decreased muscle mass in middle- to older-aged persons.[11] Weight loss associated with smoking may therefore be in part due to loss of muscle mass. It is plausible, therefore, that even though smoking may be negatively associated with body weight, it is positively related to abdominal obesity.

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Abbreviations: BMI, body mass index; CVD, cardiovascular disease; METs, metabolic equivalent tasks; MI, myocardial infarction; WHR, waist-to-hip ratio

Table 1 Principles of obesity prevention and treatment

Goal	Intervention
Prevention of weight gain (Primary prevention)	Eucaloric balanced diet 30–60 min of moderate exercise
Obesity treatment	
– induction of weight loss	Hypocaloric diet 60–120 min of moderate exercise
– weight loss maintenance (Secondary prevention)	Calorie-restricted diet* 90–120 min of vigorous exercise

*May require addition of anti-obesity medication or surgical intervention.

PRINCIPLES OF OBESITY MANAGEMENT

When considering obesity management, it is importance to make the distinction between the maintenance of a healthy body weight (primary prevention) and obesity treatment (table 1). While primary prevention of obesity merely requires the maintenance of a eucaloric balanced diet with moderate levels of physical activity, obesity treatment involves two distinct phases: (1) the induction of weight loss; and (2) weight loss maintenance (secondary prevention). Weight loss requires a negative energy balance best achieved by reducing caloric intake and moderately increasing physical activity. A daily energy deficit of around 500 kcal will translate into a weight loss of about 1 lb (0.45 kg) per week. In contrast to the weight-loss phase, which is comparatively straightforward, long term maintenance of weight loss is rarely achieved by lifestyle interventions alone.[12] As weight-loss maintenance requires life-long caloric restriction (as an ad libitum intake would immediately result in weight regain if the person reverts to his or her previous lifestyle), it is not surprising that the vast majority of individuals fail in their attempts to maintain lower body weights. We now recognise that powerful and complex biological factors are involved in the long term regulation of body weight,[13] making it difficult for individuals to maintain weight loss. Thus, the importance of pharmacological and surgical treatments of obesity lies not so much in their ability to promote weight loss (which in principle can also be achieved by dietary interventions), but rather in their ability to help patients maintain lower body weights in the long term. Therefore, anti-obesity medication should not be referred to as ''weight loss'' drugs, as their primary role is to assist in long term weight loss maintenance or secondary prevention of weight regain. It is therefore logical these agents need to be used in the long term rather than short term treatment of obesity.

CONCLUSIONS

The observation from INTERHEART that excess abdominal fat can increase cardiovascular risk at any BMI has important implications for the indication for obesity treatments. Thus, even individuals with a BMI as low as 23 kg/m^2 and a WHR of 0.95 may benefit from anti-obesity treatments, if this helps reduce their abdominal adiposity. This view of obesity treatment will radically expand the number of individuals who may need to be considered as candidates for weight loss interventions. Clinical management of obesity remains a challenge. Clearly a better understanding of the biological and lifestyle factors involved in abdominal fat deposition will increase our ability to prevent and treat abdominal adiposity, thereby reducing the cardiovascular risk associated with excess abdominal fat.

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